

Attachment 1

Amended paragraphs with corrections shown

Amend the second paragraph of Page 7 to read:

Figure 8 is a structural comparison between the native  $\beta$ -amyloid peptide and the transition state phenylalanine statine  $\beta$ -amyloid peptide analog.  $\beta$ -amyloid peptides shown correspond to amino acids 10-13 of SEQ ID NO: 3.

Amend the third paragraph of Page 7 to read:

Figure 9 is a structural comparison between the native  $\beta$ -amyloid peptide and the reduced peptide bond transition state  $\beta$ -amyloid peptide analog.  $\beta$ -amyloid peptides shown correspond to amino acids 10-13 of SEQ ID NO: 3.

Amend the fourth paragraph of Page 7 to read:

Figure 10 is a formulaic representation of the native C-terminal region of  $\beta$ -amyloid, and the phosphoramidate transition state analog of the C-terminal region of  $\beta$ -amyloid ( $A\beta_{35-43}$ ).  $\beta$ -amyloid peptides shown correspond to amino acids 1-9 of SEQ ID NO: 4.

Amend the fifth paragraph of Page 7 to read:

Figure 11 indicates the putative transition state for peptide hydrolysis by zinc peptidases, compared to the phosphonate and phosphoramidate mimics. The  $\beta$ -amyloid peptide fragments shown for the transition-state and phosphoramidate analog are HCRHNCHR (SEQ ID NO: 6). The peptide fragment shown for the phosphonate analog is HCRCHR (SEQ ID NO: 7).

Amend the sixth paragraph of Page 7 to read:

Figure 12 is a structural comparison of the native  $\beta$ -amyloid peptide and the transition state phosphoramidate  $\beta$ -amyloid peptide which has the peptide link between Gly 38 and Val 39 replaced with a phosphoramidate bond. The  $\beta$ -amyloid peptide shown corresponds to amino acid 4-7 of SEQ ID NO: 4.

In the Abstract of the Disclosure:

Please cancel the present Abstract of the Disclosure in its entirety, and replace with the following:

A7 Disclosed are bispecific antibodies comprising a first antibody binding specificity which confers the ability of the bispecific antibody to cross the blood-brain barrier, and a second antibody specificity conferring the ability of the bispecific antibody to bind to a  $\beta$ -amyloid epitope. Also disclosed are methods for inhibiting the formation of  $\beta$ -amyloid plaques in the brain of a human, or promoting the disaggregation of a preformed  $\beta$ -amyloid plaque. Such methods recite the administration of a bispecific antibody.

REMARKS

Submission of Sequence Listing

The attached paper copy of the Sequence Listing has been prepared in accordance with the provisions of 37 CFR 1.825. Instruction for amendment of the Specification to incorporate the Sequence Listing are provided above.

Also transmitted herewith is a copy of the Sequence Listing in computer readable form. As required by 37 CFR 1.821(f) and (g), Applicants' Attorney hereby states that the content of the Sequence Listing in paper form and on the computer readable form of the Sequence Listing are the same, and the submission includes no new matter.

REMARKS

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Respectfully submitted,

KM Fread

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